Editorials

The Psychobiology of War

ELSEWHERE IN THIS ISSUE is a commentary on health belief systems and the psychobiology of war by Neil J. Elgee, who is a respected internist in the Pacific Northwest. Dr Elgee has attempted what many physicians would like to see done—that is, to find a biologic or health science approach to dealing with the threat of nuclear war that would go beyond dire warnings from the medical profession of the "stupendous devastation of a nuclear war and the helplessness of the medical community to heal anything thereafter." He has obviously given this a great deal of thought and devoted much time to its study. His commentary is worth the attention of any physician who is concerned about this problem, and that should be all of us. MSMW

The Vertiginous Patient—When to Get a CT Scan of the Brain

THE VESTIBULOCEREBELLUM has been called the "fifth vestibular nucleus" because it receives primary vestibular afferents and is highly interconnected with the brainstem vestibular nuclei.[1] Lesions involving this part of the cerebellum produce vertigo, nystagmus and gait instability—symptoms that can also be produced by more benign peripheral vestibular lesions. As Dr John Hotson points out in this issue of the journal, it is important to recognize early infarction or hemorrhage involving the vestibulocerebellum because associated swelling may lead to compression of the brain stem, coma and respiratory arrest. Surgical decompression can be lifesaving.

Fortunately, most lesions involving the vestibulocerebellum also involve the lateral cerebellar hemispheres or the brain stem (or both) so that multiple associated symptoms and signs make the diagnosis of a central lesion obvious. Occasionally, however, hemorrhage or infarction is confined to the caudal midline cerebellum, and vestibular symptoms and signs predominate. As Dr Hotson notes, it is clearly inappropriate "to treat every vertiginous patient as if they have a potentially fatal stroke." It is equally inappropriate to do computed tomographic (CT) scanning of the brain on every vertiginous patient. Dr Hotson, therefore, outlines practical clinical means for separating acute benign labyrinthine disorders from strokes involving the vestibulocerebellum.

With regard to the history, I have not found a patient's description of vertigo to be particularly useful for separating central from peripheral causes. Patients typically have difficulty in deciding if they are rotating or whether the environment is rotating and in which direction the rotation is occurring. This confusion probably results from conflicting signals arriving via the vestibular, somatosensory and visual pathways, making it difficult for a patient to decide which signals to believe.

Of the clinical signs that distinguish acute benign labyrinthine disorders from lesions involving the vestibulocerebellum, I have found the nature of the nystagmus and gait instability most useful. Peripheral vestibular lesions result in unidirectional spontaneous nystagmus that is inhibited with fixation, while lesions of the cerebellum typically result in direction-changing gaze-evoked nystagmus that is poorly inhibited with fixation.[2(pp108-118)] The gait instability associated with acute lesions of the vestibulocerebellum is usually profound and patients are unable to stand without assistance. By contrast, although patients with acute peripheral vestibular lesions fall toward the side of the lesion, they can stand and walk with minimal assistance. If not clear initially, the course within the first 48 hours should distinguish between a benign peripheral and a more worrisome central disorder. The central nervous system has a remarkable ability to compensate for peripheral vestibular loss in part through activity of the vestibulocerebellum. The spontaneous nystagmus and the gait imbalance with acute peripheral vestibular lesions rapidly diminish within the first 48 hours. Lack of rapid improvement or deterioration should raise concern for a possible central lesion.

Electronystagmography (ENG) can also be useful for separating peripheral from central causes of vertigo.[2(pp125-128)] By recording spontaneous nystagmus with eyes opened in darkness or with eyes closed, the effect of fixation can be accurately assessed. Lack of suppression or an increase with fixation suggests a central lesion. Unilateral caloric hypoexcitability suggests a peripheral vestibular lesion while symmetrical hyperactive caloric responses occur with lesions of the vestibulocerebellum. Because the vestibulocerebellum is a critical center for visual-vestibular interaction, lesions involving this structure severely impair a patient's ability to modify vestibular responses with vision. Visual-vestibular interaction can be quantified with ENG by having the patient fixate on a target during the caloric test or while being rotated about the vertical axis. In normal persons or in patients with peripheral vestibular lesions the slow-phase velocity of vestibular-induced nystagmus decreases by more than 50% with fixation. By contrast, there is little change in nystagmus slow-phase velocity with fixation in patients with lesions of the vestibulocerebellum.

Which vertiginous patients require a CT scan of the brain? Dr Hotson suggests that an elderly patient or one at risk for stroke in whom findings deviate from the expected findings of a unilateral peripheral vestibular lesion requires prompt evaluation for a cerebellar in-

farction or hemorrhage. This is a reasonable formula that places proper emphasis on the clinical assessment. By way of review, the expected findings with an acute peripheral vestibular lesion are mild to moderate gait instability with a tendency to fall toward the side of the lesion, unidirectional spontaneous nystagmus that is inhibited with fixation and rapid improvement within the first 48 hours. Clearly, a small minority of vertiginous patients require a CT scan of the brain to rule out a cerebellar hemorrhage or infarction.

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REFERENCES

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Hyperinfection Syndrome in a Renal Transplant Recipient

ELSEWHERE in this issue Dr Hirschmann and co-workers discuss in detail a case of strongyloidiasis in a patient who had had a renal transplant. It is seldom that we find such an abundance of medical wit and philosophy dispensed in equal measure with diagnostic acumen in a clinicopathologic conference. In addition to reaching a correct diagnosis, the discussant covered very well virtually all of the important clinical aspects of strongyloidiasis.

Before expanding a bit on some features of strongyloidiasis, it might be appropriate to at least mention another parasite, namely *Toxoplasma gondii,* that might have been considered in association with such complicated illness in an immunodeficient host. While toxoplasmosis can cause skin lesions and pulmonary involvement, the skin lesions are likely to be vasculitic and definitely not pyogenic in nature. The pulmonary lesions are also much less prominent and nondescript in nature. In addition, central nervous system invasion is often a terminal feature of toxoplasmosis in an immunocompromised patient.

Bringing the discussion back to strongyloidiasis, one of the first points to emphasize is that exposure to this parasite does not require travel to or residence in the tropics. While strongyloidiasis is much more common in certain geographic areas, such as in Southeast Asia, it also occurs in the United States. In fact, the parasite is better adapted to transmission in an unsanitary environment than many other intestinal helminths by virtue of its rapid transformation to the infective stage. This may help explain why infection with *Strongyloides stercoralis* is seen in mental institutions and in asylums where hygienic conditions are more often compromised.[1]

Incidentally, the Australian experience referred to by Dr Hirschmann in World War II soldiers interred in Japanese prisoner of war camps was also reported from England.[2] A similar experience has been noted also in the United States military during World War II by Pelletier.[3] This documentation indicates that parasites from some areas of the world are capable of persisting indefinitely in a human host, presumably by a low level of internal autoinfection, and this raises an interesting question as to why immunity in this infection is not more effective.

The pulmonary symptoms present in the case described in this issue deserve a few additional comments. First, the adult female parasite may sometimes be found in the lungs as well as in the upper small bowel, so that larvae found in sputum may be produced there and not be simply in transit through the lungs. In fact, in this particular case the larvae shown in Figure 2 of the Specialty Conference in this issue are compatible with larvae transiting the lungs, at or beyond the filariform stage, rather than the rhabditiform stage. If authentic rhabditiform larvae are seen in the sputum this is an indication that female worms are present and the infection may be somewhat more difficult to treat than if the infection is only intestinal.

Another point with regard to pulmonary involvement is that in some patients asthmatic symptoms develop with *Strongyloides* infection. The wheezing and dyspnea described in this patient may have been due to bronchial constriction on an allergic basis and not simply to a Gram-negative pneumonia. Occasionally, the prominent asthmatic features of *Strongyloides* infections seen in some patients may be reminiscent of tropical pulmonary eosinophilia with diffuse pulmonary infiltrates bilaterally, leukocytosis and a striking degree of eosinophilia. Incidentally, eosinophilia is very commonly seen in patients infected with *S stercoralis,* although usually a patient has an eosinophilia on the order of 10% to 25%. In this case, of course, eosinophilia may have been suppressed by the large doses of steroids or, as pointed out by Dr Hirschmann, some patients with overwhelming strongyloidiasis, even when not receiving steroids, do not mount a peripheral eosinophil response. A recently described enzyme-linked immunosorbent assay test for antibody to larval antigen may be helpful in diagnosis because larval excretion can be irregular and laboratory personnel may be inexperienced in examining stool or sputum specimens for larvae.[4]

It is interesting that diffuse pulmonary infiltrates are frequently described in fatal cases of strongyloidiasis reported in the literature. The pathologic description in these cases often comments on the hemorrhagic lungs. This is of some interest because in recent work with a monkey model of overwhelming strongyloidiasis it was reported that hemorrhagic lungs are an invariable feature of fatal overwhelming *Strongyloides* infection in the patas monkey.[5]

Finally, I would take exception with one of the points made in the discussion of the case that the finding of filariform larvae in a stool or sputum specimen makes a diagnosis of overwhelming strongyloidiasis conclusive. Certainly the finding of filariform larvae in a sputum specimen indicates that larvae are transiting